Treatment of the Obstructive Sleep Apnea Syndrome

ROBERT V. WIGGINS, MD, *and* WOLFGANG W. SCHMIDT-NOWARA, MD, *Albuquerque*

The obstructive sleep apnea syndrome is a disorder of sleep and breathing that is being recognized with increasing frequency. The pathophysiologic consequences range from mild sleepiness to life-threatening cardiovascular and respiratory decompensation. The primary forms of treatment are directed at modifying the upper airway with either an operation or continuous positive airway pressure. Aside from tracheostomy, which is virtually always successful, other forms of treatment have met with varying results. Ancillary therapy, including oxygen, weight loss and drugs, is often helpful but seldom curative. Follow-up sleep studies are necessary to evaluate the effectiveness of treatment. Selecting therapy for a patient with obstructive sleep apnea requires a comprehensive evaluation including polysomnography, special examinations of the upper airway and assessing the cardiopulmonary status. Therapy is based on the severity of disease and must be tailored to each patient.

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Since its first description two decades ago, the obstructive sleep apnea syndrome has become the subject of extensive investigation and clinical activity. With progress in understanding the pathophysiology, various surgical and nonsurgical treatments have been developed and are available to clinicians treating sleep apnea. Most reviews of this subject have focused on the pathophysiology, but several reviews of treatment have appeared recently.[1-4] In this article we focus on treatment and particularly on clinical reports that permit a critical appraisal of the efficacy of varied treatments. The review will focus on the condition as it occurs in adults because most of the literature deals with this age group.

In view of the newness of the current concepts of sleep apnea, it is not surprising that the indications and efficacy of various treatments remain controversial. The very number of available choices testifies to the lack of a universally acceptable solution. Most treatments are effective only to varying degrees in selected groups of patients. Furthermore, most therapies represent a significant intervention in terms of patient involvement, risk of complication or cost. Thus, selecting the best therapy for each patient requires an analysis of risk versus benefit. Unfortunately, relatively few systematic or controlled studies have been conducted that provide the necessary information for this analysis. The emphasis of this review will be on information that facilitates this risk-benefit analysis.

Clinical Manifestations

The clinical manifestations of obstructive sleep apnea are related to upper airway obstruction, sleep disruption and the respiratory and cardiovascular consequences of disordered breathing.[5,6] Snoring is universally present when both patients and families are carefully interviewed. Less frequent indications of obstruction include struggling for breath and choking, with repeated arousals. The consequences of sleep disruption are similar to those of sleep deprivation, with pathologic sleepiness as the primary symptom. Other symptoms include fatigue, personality change, depression, impotence and automatic behavior. The respiratory consequences are related to the extent of hypoxemia and hypercapnia that develop with the hypoventilatory episodes as well as to their frequency. Thus, more advanced cases are associated with pulmonary hypertension, cor pulmonale, chronic carbon dioxide retention and polycythemia. Most patients, however, do not manifest these disturbances, presumably because their ventilation during wakefulness is normal enough to prevent these complications of chronic hypoxia. The cardiovascular consequences are systemic hypertension and cardiac arrhythmias during sleep. Sudden death has occurred in patients with sleep apnea.[6]

Pathophysiology

The pathophysiology of the obstructive sleep apnea syndrome has been reviewed recently and will not be considered in detail.[7-9] For orientation, however, a brief summary is necessary.

The sleep apnea syndrome is a group of clinical conditions characterized by abnormal breathing in sleep and sleep fragmentation. In patients with the obstructive sleep apnea syndrome, obstruction of the upper airway during sleep results in intermittent hypoventilation or overt apneas. The resulting hypoxemia and hypercapnia produce arousal with restoration of the airway and the resumption of breathing. When this sequence of events occurs often enough—typically hundreds of times each night—the combined effects on sleep and cardiopulmonary function produce the morbidity associated with this condition. In a small number of patients, those with predominantly central apneas, an absent respiratory effort rather than obstruction appears to be the principal cause of apneas. Recent investigation suggests, however, that many of these patients also have associated upper airway dysfunction and benefit from measures that improve airway patency.[10,11]

From the Division of Otolaryngology, Departments of Surgery (Dr Wiggins) and Medicine (Dr Schmidt-Nowara), Center for Otolaryngology and Communicative Disorders, University of New Mexico School of Medicine, Albuquerque.

Reprint requests to Robert V. Wiggins, MD, Department of Surgery, ENT Division, University of New Mexico, Albuquerque, NM 87131.

ABBREVIATIONS USED IN TEXT

CPAP = continuous positive airway pressure
REM = rapid eye movement
SI = severity index

The causes of upper airway obstruction in the obstructive sleep apnea syndrome are complex and controversial. The site of the obstruction has been identified as the oropharynx and nasopharynx.[12-14] Gross anatomic factors play a major role in cases associated with tonsillar hypertrophy, macroglossia and micrognathia.[15-17] Even in patients without an obvious pathologic disorder of the upper airway, measurements by computed tomography identify substantial narrowing of the oropharyngeal airway compared with normal persons.[18] Functional abnormalities have been identified, including increased upper airway resistance and increased compliance of the upper airway.[19,20] Reductions in respiratory drive have been identified by altered breath timing and by decreased diaphragmatic and upper airway muscle activity, and although their cause is uncertain, they may be significant elements in the pathogenesis of obstruction and apneas.[21-23]

Thus, a variety of mechanisms contribute to the upper airway obstruction in this disorder, but identifying the principal cause in an individual patient is difficult. When surgical treatment is contemplated, the anatomy of the upper airway and the precise site of obstruction become a major consideration. Of the many investigational techniques used to assess the upper airway, nasopharyngeal endoscopy, lateral cephalometrograms and computed tomography appear to be clinically practical and may have some value for guiding surgical decisions.[12,14,24-27] Physical examination occasionally reveals gross abnormalities of the facial skeleton or oropharynx, but in most patients the results of an examination are not a reliable guide to therapy or a predictor of outcome.[28,29]

Treatment

The treatment of sleep apnea has focused on correcting the various physiologic derangements just discussed. For the purpose of this review, treatment will be classified according to therapies directed at airway patency, including surgical procedures of the upper airway, prosthetic devices and continuous positive airway pressure (CPAP), and ancillary therapies directed at disturbances of gas exchange and abnormalities of respiratory rhythm (Table 1).

Surgery of the Upper Airway

Surgical treatment of obstructive sleep apnea appeals to patients and physicians alike as a permanent "fix" for a complicated and troubling disorder. As the pathophysiology of the obstructive sleep apnea syndrome has become defined, various surgical treatments have been developed. The relative efficacy of individual procedures is somewhat difficult to assess from the literature. Central to this problem is the broad spectrum of severity of this disease and the lack of a clear definition of what constitutes a "cure." An operation is a powerful placebo, and thus subjective improvement alone cannot be accepted as proof of efficacy. Such a conclusion must be supported by objective data, including, at a minimum, a complete postoperative polysomnogram.

Tracheostomy. Tracheostomy was the first efficacious procedure described for sleep apnea and remains the procedure against which all others should be measured.[30,31] Guilleminault and co-workers reported a series of 50 patients with severe obstructive sleep apnea who underwent tracheostomy.[32] Before the procedure, all but one patient had an oxygen desaturation to 30% or lower and all had an apnea index of 65 or more apneas per hour. Following the operation, the apnea index dropped to 5 or less and the lowest desaturation was 82%. Of these patients, 47 were employed or in school within three months postoperatively. Weitzman and colleagues showed more consolidated sleep with fewer arousals in ten sleep apnea patients following tracheostomy.[33] Obstructive and mixed apneas were eliminated. Complications of tracheostomy, however, though not severe, are frequent. Guilleminault and associates reported that 42% of their patients experienced recurrent low-grade infections and airway granulations.[32] Conway and co-workers noted similar problems, including stomal stenosis, in 8 of 11 patients.[34] Such difficulties may be lessened with the skin-flap tracheostomy as described by Fee and Ward.[35]

TABLE 1.—*Treatment of Obstructive Sleep Apnea*

Modification of the upper airway
Tracheostomy
Uvulopalatopharyngoplasty
Nasal operation
Orthognathic operation
Nasal CPAP
Prosthetic devices
Adjunctive therapy
Weight reduction
Oxygen
Pharmacotherapy
Position

CPAP=continuous positive airway pressure

Clearly, tracheostomy cures sleep apnea. Just as obvious, however, are the medical and social problems associated with a permanent tracheostomy, and these have stimulated the search for alternative therapies.

Uvulopalatopharyngoplasty. Other surgical procedures for the obstructive sleep apnea syndrome involve enlarging or expanding the upper airway, thereby preventing its collapse. The selection of one or more of these procedures should be based on objective findings documenting the level or levels at which the obstruction occurs. A simple procedure, such as adenotonsillectomy, may be all that is necessary in selected patients. Eliaschar and colleagues showed a significant reduction in the number of obstructive apneas in children undergoing tonsillectomy for obstructive sleep apnea.[36] Orr and Martin showed that tonsillectomy in adults with even relatively mild tonsillar enlargement alleviates sleep apnea.[15]

Uvulopalatopharyngoplasty is probably at present the most frequently done procedure for sleep apnea. Initially described in the American literature by Fujita and associates, this procedure involves shortening the soft palate, amputating the uvula and removing redundant lateral and posterior pharyngeal wall mucosa from the oropharynx.[37] Of their initial 12 patients, 11 described a subjective abatement of daytime sleepiness. Sleep indices, including the number of apneas, oxygen saturation and the number of arousals, were significantly improved for the group as a whole. Three patients, however, were essentially unchanged, one patient was worse and three others had improvement but continued to show evidence of residual sleep disturbance when evaluated by poly-

somnography. Thus, severe sleep apnea may be converted to a milder disease. Simmons and co-workers reported that 17 of 20 patients who underwent uvulopalatopharyngoplasty reported symptomatic improvement.[28] Objective improvement in sleep indicators, including the apnea index and oxygen saturation, was exhibited by only 50% of this group. Katsantonis and associates evaluated their results by measuring the "severity index (SI)"—that is, the number of obstructive events per hour resulting in oxygen desaturation below 85%.[29] Nearly two thirds of their series of 31 patients showed a reduction in the "SI" of better than 50%. While all authors report a substantial reduction in snoring as well as symptomatic improvement, there is no clear agreement on what constitutes a cure of sleep apnea or, for that matter, what is meant by significant improvement. Using rigorous guidelines for decannulation, Borowiecki and Sassin were unable to decannulate ten of ten patients who underwent uvulopalatopharyngoplasty in conjunction with tracheostomy.[38] The likelihood of a successful result may improve with careful patient selection based on special preoperative studies.

Using the Mueller maneuver, Sher and colleagues evaluated patients who had fiber-optic nasopharyngoscopy to determine the anatomic site of airway collapse.[24] Patients showing collapse at the soft palate received uvulopalatopharyngoplasty, and 87% had significant improvement. Walsh and Katsantonis evaluated 16 patients with somnofluoroscopy before and after uvulopalatopharyngoplasty.[39] Those patients whose airways were narrowest in the mid and upper oropharynx tended to respond more favorably.

Nasal surgery. Nasal operations have been shown to diminish sleep apnea in selected patients.[40-42] In general, these are patients with obvious obstruction due to deformity of the nasal septum, or polyps, and relatively mild sleep apnea. Patients with moderate to severe sleep apnea are not as likely to benefit from a nasal operation alone, though it may be a useful adjunctive procedure. Nasal obstruction has been related to excessive daytime sleepiness and arousals, periodic breathing and hypopneas during sleep.[43] Following a nasal procedure, 12 of 14 patients had improvement, evidenced by more consolidated sleep and fewer arousals.

Orthognathic surgery. Mandibular deficiency has been associated with sleep apnea. Bear and Priest reported the case of one patient with obvious mandibular retrognathism and sleep apnea who was cured by surgical advancement of the mandible.[44] More recently, using lateral cephalometric skull radiographs, Guilleminault and co-workers have shown subtle facial skeletal abnormalities in patients with sleep apnea.[25] In their series, patients had a narrow posterior airway space and a low-lying hyoid bone. Accordingly, they have devised a procedure that advances the anterior-inferior mandibular arch, together with the genioglossus muscle, and suspends the hyoid bone. In their series of five patients in which uvulopalatopharyngoplasty failed for three, four of five showed significant subjective and objective improvement.[45] Carrying this concept a step further, the authors did maxillary, mandibular and hyoid advancements in a series of nine patients.[46] Most of these patients showed some mandibular deficiency radiographically, but two had normal facial skeletal relationships. All patients showed significant symptomatic improvement and pronounced improvement on postoperative polysomnography.

Miscellaneous surgical procedures. The problem of hypopharyngeal collapse has been addressed by Patton and Thawley using a procedure that involves sectioning the hyoid bone and expanding it using a stainless-steel brace.[47] Their work in dogs has shown that the hypopharynx is enlarged and is less likely to collapse with this procedure, but the experience in patients is too limited to assess at this time. Fujita and associates have recently presented another approach to the problem of hypopharyngeal collapse.[48] Tongue reduction by midline laser glossectomy was done in selected patients with good results. This procedure, too, requires further study.

Surgical treatment has also been used to reduce weight in morbidly obese patients as the principal therapy for the obstructive sleep apnea syndrome.[49,50] Weight loss as therapy will be discussed in a subsequent section.

Prosthetic Devices

The use of prosthetic devices in the treatment of the obstructive sleep apnea syndrome is based on the same principles used in surgical treatment. The two areas of focus anatomically are the nasopharyngeal inlet and the posterior tongue. Collapse at the level of the soft palate has been managed with a rubber nasopharyngeal airway, commonly used for postanesthetic airway management.[51,52] The mandible may be temporarily advanced by inserting an acrylic dental appliance before sleep.[53,54] Cartwright and Samuelson have used a tongue-retaining device to hold the tongue in a more anterior position during sleep.[55] This device has also been used successfully in some patients who have not had improvement following uvulopalatopharyngoplasty.[56] Nasal breathing is obligatory with both the dental appliance and the tongue-retaining device, and thus the nasal airway must be adequate for them to be of benefit. The disadvantage of prosthetic devices is the need to insert them nightly and the discomfort associated with wearing them. Experience with all of these devices is limited, but they may prove to be useful in selected patients.

Nasal CPAP

Successful treatment of sleep apnea by using continuous positive airway pressure (CPAP) was first reported by Sullivan and colleagues in 1981.[57] Subsequent reports have shown this therapy to be almost uniformly successful in a broad spectrum of patients with the obstructive sleep apnea syndrome.[58-62] Nasal CPAP appears to work as a "pneumatic splint" of the upper airway, the positive pressure preventing passive collapse of soft tissues during inspiration. Apneas and hypopneas are eliminated, and consolidated sleep returns. Periodic oxygen desaturation is corrected, though hypoxemia may persist in patients having alveolar hypoventilation or chronic lung disease, and such patients may require oxygen supplementation. Symptomatic improvement is dramatic, with pathologic daytime sleepiness reduced or eliminated, a result recently confirmed by multiple sleep latency tests.[63]

Despite the elegant simplicity of the concept, CPAP has not been easy to implement, and patient compliance has been a problem. Applying pressure through the nose appears to be the most suitable for patient comfort and ease of application. Sullivan and colleagues initially used intranasal tubes, and other investigators have used a similar design.[57,60,62] Most investigators, however, use a mask held against the face with sufficient pressure to prevent air leak. Commercial versions of this technique are now available. Initially recommended for the short-term relief of sleep apnea, nasal CPAP has been used for long-term care. Not all patients accept this therapy,

however. In a series of 50 patients treated by Sullivan and co-workers, 35 selected nasal CPAP for long-term use.[59] Recent reports from this country indicate that patient acceptance may be as low as 60%.[64,65] The reasons for dissatisfaction include irritation of skin and eyes, the latter from air leaks around the mask, nasal congestion and sore throat and the inability to tolerate the noise. Thus, implementing nasal CPAP, particularly for long-term use, requires careful patient selection, training and follow-up.

Ancillary Therapies

Weight reduction. Weight loss should be emphasized in the treatment of sleep apnea. Burwell's classic description of the pickwickian syndrome documents the beneficial effect of relatively modest weight loss on sleepiness and on cardiopulmonary function.[66] Early reports of case series of sleep apnea refer to the benefits of weight loss but without systematic observations.[5] More recently a well-controlled study of 16 patients showed fewer apneas and reduced oxygen desaturation along with lessened daytime sleepiness in patients who lost about 20 kg (45 lb) when compared with a similar group of patients who did not lose weight.[67] Several studies in patients with morbid obesity have shown improved oxygen saturation and usually reduced apnea frequency after weight loss produced by a weight-reducing operation.[49,50,68] Thus weight loss can be beneficial in patients with the obstructive sleep apnea syndrome and obesity of a moderate to a severe degree. Subjective improvement usually accompanies these objective changes, but some disturbance of sleep and breathing usually remains.

Oxygen therapy. The role of oxygen in the treatment of sleep apnea has been surprisingly controversial. The argument against oxygen therapy is based on the belief that supplemental oxygen will delay arousal from apnea. Motta and Guilleminault presented this argument based on limited observations in three patients with obstructive apneas and one with central apneas in whom oxygen administration approximately doubled the duration of apneas during non-rapid-eye-movement (REM) sleep and respiratory acidosis was made worse.[69] Martin and associates subsequently reported the cases of eight patients with obstructive apnea in whom oxygen administration consistently reduced the severity of oxygen desaturation.[70] In five patients, long-term oxygen therapy substantially reduced the number of apneas. Smith and co-workers reported the cases of 15 patients who improved or did not worsen with oxygen when apnea frequency, duration and degree of desaturation were examined.[71] The same investigators subsequently reported that long-term—that is, one-month—oxygen therapy did not correct pathologic sleepiness despite improvement of nocturnal oxygenation.[72]

The combined experience of these reports suggests that in most patients with obstructive apnea, oxygen can be used safely and will produce beneficial effects on respiration. In some patients, perhaps those with high-grade upper airway obstruction, apnea duration may be prolonged. The concern that such prolongation may produce a worse outcome is not supported by published data. It should be noted, however, that the effect of oxygen use has been studied carefully in only a small number of patients, with considerable individual variation in each investigation. We believe that the dangers of profound hypoxemia are greater than the theoretic concerns about prolonged apnea and that oxygen therapy should not be withheld when hypoxemia is profound, particularly when secondary effects such as bradycardia, pulmonary hypertension or erythrocytosis are present. The effect, however, of oxygen therapy on a patient with high-grade airway obstruction or chronic respiratory acidosis, or both, should be monitored with oximetry or complete polysomnography.

Pharmacotherapy. Several drugs have been advocated to use for treatment, but their value is limited in patients with the obstructive sleep apnea syndrome. The most effective and best studied drug is protriptyline hydrochloride, a nonsedating tricyclic agent whose use was found to alleviate abnormal breathing during sleep and to lessen daytime somnolence in four patients with narcolepsy and sleep apnea.[73] In a subsequent study of patients with varying degrees of obstructive sleep apnea, 12 of 14 patients had symptomatic and objective improvement with doses ranging from 2.5 to 25 mg a night.[74] Anticholinergic side effects were frequent, however, resulting in alternative therapy in 50% of patients. In a double-blind crossover study of six patients, the use of protriptyline lessened daytime sleepiness without affecting overall apnea frequency and duration.[75] Smith and associates showed a reduction in apnea frequency and oxygen desaturation during non-REM sleep, as well as a reduction in REM sleep.[76] Of their 12 patients, 10 were subjectively improved, and although side effects were frequent, 11 of 12 tolerated prolonged therapy. The beneficial effect of using protriptyline has been attributed to its suppression of REM sleep and also to a preferential stimulation of upper airway muscle tone.[75,77] Thus, protriptyline offers reasonable prospects for symptomatic improvement and may reduce the degree of oxygen desaturation, but frequent side effects and incomplete efficacy limit its value.

Medroxyprogesterone acetate has been used because of its respiratory stimulant effect and its proved benefit in the obesity-hypoventilation syndrome.[78] In seven patients with the sleep apnea syndrome, however, the use of progesterone did not alter apnea frequency, although oxygenation improved.[79] In another study of eight patients with the obesity-hypoventilation syndrome, improvement occurred in four patients with CO_2 retention but not in the patients with a normal partial pressure of arterial CO_2.[80] Therefore, medroxyprogesterone has limited value in the treatment of obstructive sleep apnea, but it may be effective when the obesity-hypoventilation syndrome with chronic hypercapnia is present.

Acetazolamide, a carbonic anhydrase inhibitor, stimulates respiration by producing a metabolic acidosis. In a group of patients with predominantly central sleep apnea, the use of this drug reduced the number of apneas and improved symptoms.[81] In several cases with mild obstructive apnea, however, acetazolamide administration was associated with more frequent obstructive apneas of longer duration.[82] Thus, the drug is probably not useful in patients with the obstructive sleep apnea syndrome, and we do not use it in these patients.

Miscellaneous adjunctive therapies. Several other adjunctive therapies may be useful in selected patients. The sleep position may affect sleep apnea frequency, with the supine posture resulting in the highest apnea frequency. In some patients, apneas occur only in this position. If these patients can be trained to sleep on their side, sleep and breathing may be substantially improved,[83] but the feasibility of this approach for long-term care remains to be proved. Another report suggests that moving from the recumbent to a sitting posture can bring immediate improvement.[84] Severely affected patients often sleep in a chair because of this side

effect, but the benefit is marginal because the improvement of sleep and breathing is incomplete. Medical therapy for nasal obstruction due to allergic or vasomotor rhinitis may be helpful, but its effects in patients with the sleep apnea syndrome has not been studied.

Evaluation

Selecting one or several of the above therapies hinges on a careful evaluation of patients. The probability of diagnosis, the degree of disability, etiologic factors and the potential for various therapies should all be considered in doing a history and physical examination, sleep studies and adjunctive tests (Figure 1). The history should not only investigate symptoms of disturbed breathing during sleep and its consequences, but should also develop the relationship of these symptoms to age, weight, sleeping position, the use of alcohol or medications and any other medical history. Because patients may be unaware of the events at night and may say they are not sleepy during the day, an interview with a spouse or some other observer is important. In our experience, however, the obstructive sleep apnea syndrome usually produces symptoms that by themselves are not specific but in the aggregate produce a strong clinical impression of the diagnosis. Snoring, arousals from sleep with choking or gasping, spouse-observed apneas and pathologic sleepiness in passive or monotonous circumstances are usually present and occur more frequently than in normal subjects. Carefully assessing pathologic sleepiness is essential, as this is the principal consequence of obstructive sleep apnea, and we use this symptom to classify the severity of the disease. Occasionally, the sleep apnea syndrome produces a complaint of insomnia, but in our experience, this complaint is usually accompanied by symptoms of abnormal breathing during sleep.

Physical examination of a patient should include a thorough general examination. Measurement of height and weight, blood pressure, careful assessment of lung and cardiac function and an assessment of the neuromuscular system are all relevant to the diagnosis of this disease. In addition,

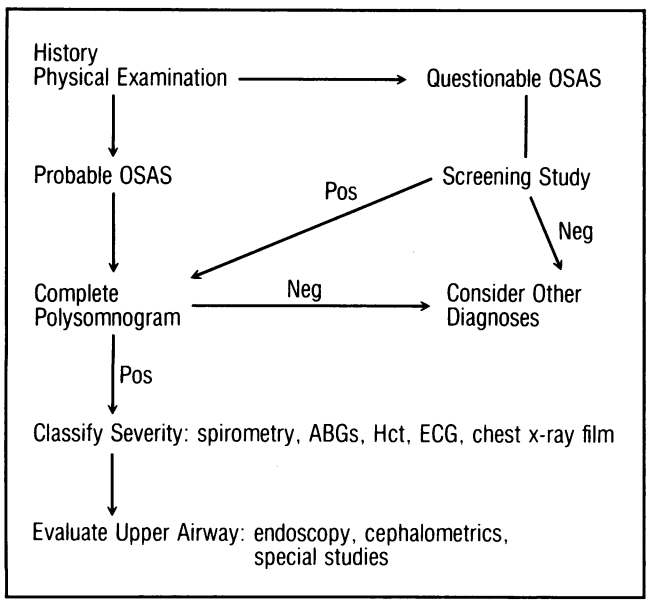


Figure 1.—The schema shows the process for evaluating patients for the obstructive sleep apnea syndrome (OSAS). ABGs = arterial blood gas determinations, ECG = electrocardiogram, Hct = hematocrit

examination of the upper airway is critical. The facial appearance often provides a clue to a maxillary or mandibular deficiency. The mouth and throat should be examined for a gross pathologic condition, including malocclusion, tonsillar hypertrophy, a long uvula and redundant pharyngeal mucosal folds. Special consideration should be given to the relative size of the oral cavity and tongue. The position of the soft palate relative to the posterior pharyngeal wall may give some indication of the potential for occlusion. Although most patients with the obstructive sleep apnea syndrome do not have striking anatomic abnormalities, the throat often appears "crowded" and the oropharyngeal airway appears small.

The next step in the evaluation is an objective evaluation of breathing in sleep. If by history the probability of the sleep apnea syndrome is low, we recommend a screening test using one of several home monitors or, for inpatients, an all-night recording of oxygen saturation during sleep. If a reasonable probability of the syndrome can be established by the initial interview, however, or if the screening test is positive, a complete polysomnogram should be done. Polysomnography is the current standard for diagnosis because it defines the problem in all its dimensions: sleep disturbance, respiratory disturbance including apneas and hypopneas, oxygen desaturation, cardiac arrhythmia and sleep position.

When the sleep apnea syndrome is confirmed, more detailed examinations are necessary to assist the selection of therapy. A fiber-optic examination of the airway is helpful in assessing the site of obstruction and may help predict those patients most likely to respond favorably to surgical treatment.[24] We have patients execute the Mueller maneuver, although interpreting this maneuver is difficult. If the facial appearance suggests a skeletal abnormality, a lateral cephalometric radiograph is done.

Additional examinations include an assessment of cardiopulmonary function. Spirometry, arterial blood gas determinations, hemoglobin concentration, electrocardiography and chest x-ray films all serve to evaluate the presence of coexistent lung disease, respiratory failure and cor pulmonale. A 24-hour Holter monitor may be appropriate if polysomnography has identified cardiac arrhythmias other than the typical sinus variation. In view of the frequency of thyroid disease in the general population and the high prevalence of sleep apnea in hypothyroid patients,[85] a routine assessment for hypothyroidism and treatment if results are abnormal appear justified. Consideration of other endocrine disorders should be based on physical findings.

Classification—Therapeutic Recommendations

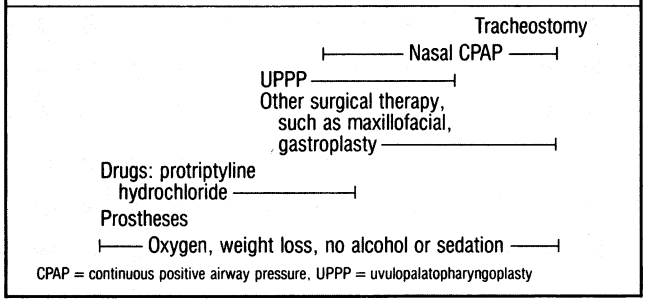
After a complete examination, it is useful to classify the severity of disease by a combination of clinical and polysomnographic criteria (Table 2). In addition to conventional polygraphic indicators of breathing in sleep, we consider the extent of sleep fragmentation—that is, the frequency of periodic respiration-related arousals—and periodic breathing, defined by stereotypic periodic desaturations on a compressed recording of oxygen saturation. The criteria for classification are arbitrary but correspond approximately to current symptoms and disability. Treatment is then based on the severity of disease (Table 3). The importance of tailoring the treatment to each patient cannot be overemphasized. Factors such as age and other medical conditions influence the choice of therapy. A frail, elderly person may present an unacceptable surgical risk. On the other hand, a younger patient may find long-term

TABLE 2.—*Obstructive Sleep Apnea Syndrome: Classification of Severity*

Variable	*Mild*	*Moderate*	*Severe*
Sleep Data			
Sleep	Mostly normal; <25% of sleep fragmented	Some consolidated sleep present, no stage 3 or 4	Totally fragmented
Breathing, % periodic	<50	50-90	>90
Apneas			
Number/h	<20	<40	>40
Duration, s	<20	<30	>30
O_2 saturation, %			
Mean	>90	>85	<85
Lowest	>80	CO_2 partial pressure normal; no heart failure	Elevated CO_2 partial pressure, hematocrit; cor pulmonale, heart failure

CO_2 = carbon dioxide, ECG = electrocardiogram, O_2 = oxygen

TABLE 3.—*Therapy for the Obstructive Sleep Apnea Syndrome*



CPAP = continuous positive airway pressure, UPPP = uvulopalatopharyngoplasty

nasal CPAP therapy unacceptable and may prefer a surgical alternative.

All patients with sleep apnea are instructed to avoid using respiratory depressants such as alcohol, narcotics and sedatives. Avoidance of sedatives is particularly important with surgical treatment. We do not administer sedatives preoperatively until a patient is in the operating suite, and after an operation we observe the patient in the intensive care unit for the first 24 hours.

Patients with severe disease show extreme pathologic sleepiness and respiratory failure. Because they are at risk for sudden death,[6] the choice of treatment should be nasal CPAP or tracheostomy, the two treatments that have the greatest likelihood of immediate success. An ancillary operation such as uvulopalatopharyngoplasty or other surgical procedures may be included along with the primary forms of therapy and, if successful, may eventually allow the patient to have a tracheostomy closed or nasal CPAP discontinued. Significant weight loss in obese patients may likewise eliminate the need for long-term tracheostomy or nasal CPAP, after some time.

Patients with moderate obstructive sleep apnea complain of sleepiness but do not have respiratory failure and their disability is limited. The long-term prognosis of these patients is unclear, and treatment must be tailored to each person. Tracheostomy is usually not justified medically or acceptable to the patient. Assessing the upper airway may reveal obstruction that can be corrected surgically. The biggest problem, however, is the common situation of a moderately impaired patient whose airway may not be obviously correctable. Uvulopalatopharyngoplasty may be offered but only as a procedure with a significant chance of failure. Newer surgical procedures are not sufficiently evaluated to define their role. As a result, nonsurgical therapy is a major consideration. Nasal CPAP is likely to be efficacious but will not be acceptable to all patients. The use of protriptyline, oxygen therapy and prosthetic devices should be considered. Weight loss should be a part of every regimen.

Mild sleep apnea may be quite subtle. Sleepiness is minimal and medical complications are not present. As with moderate sleep apnea, the prognosis is not known, and treatment must be individualized. Surgical treatment is generally not recommended unless there are other associated problems such as nasal obstruction or favorable findings such as enlarged tonsils. Weight reduction is appropriate in most patients. The use of protriptyline or other pharmacologic therapy may be helpful.

All patients require follow-up. Because therapies are not always effective, every treated patient must be reevaluated to assure that the desired clinical result has been achieved. In most cases, this evaluation will require some objective assessment of breathing in sleep. Because the clinical experience with sleep apnea is short and the experience with many therapies is shorter and because the prognosis for patients cannot be predicted with certainty, all should be reevaluated periodically to identify progression of disease or relapses after successful treatment.

Conclusion

The obstructive sleep apnea syndrome is a disorder characterized by abnormal breathing during sleep and manifests a wide range of severity. The treatment selected must be based on a thorough evaluation of the patient and assessment of the severity of disease and then tailored to the individual patient according to the relative risks and benefits of the therapy. The efficacy of the therapy chosen should be monitored both subjectively and objectively. Controlled prospective clinical trials are needed to better evaluate the various treatment options available.

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Book Review

The Western Journal of Medicine *does not review all books sent by publishers, although information about new books received is printed elsewhere in the journal as space permits. Prices quoted are those given by the publishers.*

Trauma in Children

Edited by Randall E. Marcus, MD, Coordinator of Trauma Services and Assistant Professor of Orthopedic Surgery, Case Western Reserve University School of Medicine, and Attending Orthopedic Surgeon, Rainbow Babies and Children's Hospital, Cleveland. Aspen Publishers, Inc, 1600 Research Blvd, Rockville, MD 20850, 1986. 265 pages, $54.

Trauma in Children is a 265 page, one volume book edited by Randall Marcus with 20 other contributors. The chapters discuss trauma in children by anatomic regions including the head and central nervous system, chest, abdomen, genito-urinary region, hand, spine and face. There are also chapters on fractures, soft tissue injuries and complications of musculoskeletal injuries, as well as introductory chapters on initial management and general principles of trauma management in children. The last two chapters of the book address psychological and legal implications of trauma.

Each chapter is an independent discussion of a topic by professionals with personal experience in the field. In general, each chapter provides an overview with more extensive discussions of the common or serious injuries. The depth of discussion varies among chapters but generally is fairly superficial. Someone desiring to understand current management of a specific fracture, for instance, would find a much more complete discussion of the topic in a specialty text.

There are some distracting editorial shortcomings such as mislabeled figures. There are very few pictorial examples of long-term followup or even final results of fracture treatment, an important consideration in pediatric fractures. Omissions occur in other chapters. For instance, there is no mention of child abuse in the chapter "Legal Considerations in the Treatment of Trauma in Children."

The specific topic of trauma in children has not been well addressed previously in book form in this country. This book is a good survey of pediatric trauma and crosses the conventional boundaries of medicine. It would be of value to someone who occasionally deals in pediatric trauma who would like to become familiar with some of the current concepts. It would also be of value to the trauma subspecialist to introduce the principles of treatment of pediatric trauma in other disciplines or anatomic regions.

THOMAS A. DeCOSTER, MD
Department of Orthopaedics and Rehabilitation
University of New Mexico Medical Center
Albuquerque